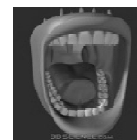


Diseases of the mouth



Stomatitis : Inflammation of oral cavity

1. Bacterial (vincent's infection) :	✓ c/p : - caused by fusiform bacteria & spirochetes - Halitosis - Ulcers on the gum, palate, lips and the inner aspects of cheeks ✓ ttt. : Metronidazole or penicillin
2. Viral stomatitis:	✓ c/p: - Herpes simplex may cause herpes labialis in normal persons & severe form in impaired immunity. - Coxsackie virus causes herpangina with acute pharyngitis, ulcers of the soft palate and pharyngeal mucosa. ✓ ttt: anti viral in severe cases
3. candidiasis (Monillasis):	✓ c/p : <u>Organism</u>: fungus candida albicans - White patches on the tongue and buccal mucosa . ✓ ttt : Lozenges or suspension of nystatin.& Systemic antifungal in severe infection.
4. Nutritional :	✓ c/p : - deficiency of niacin, riboflavin, folic acid, iron and vitamin B12 - the tongue is red and painful because of atrophy of the papillae. ✓ ttt: of the cause
5. Aphthous ulcers:	✓ c/p : Affecting 2% of the population - Emotional stress & Premenstrual phase. - Multiple shallow rounded, painful ulcer - The ulcers are recurrent at intervals of days to a few months. - minor < 10mm & major > 10mm ttt : ✓ Hydrocortisone hemisuccinate lozenges. ✓ Topical anesthetics ✓ Colchicine

DD of mouth ulcers:-

1. **Skin diseases:-** Erythema multiforme - Lichen planus - Alpha methyl dopa.
2. **Drugs:-** Antimalarials - Penicillamine
3. **Systemic disorders:-** Inflammatory bowel disease - SLE
4. **Malignant tumors of the mouth:** Squamous cell carcinoma.

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Diseases of the tongue

- ☒ **Glositis**: Glositis is a red, smooth, sore tongue seen in B12, folate or iron deficiency
- ☒ **Leukoplakia**: Chronic lesion (White, firm smooth patches), usually starts at the side of the tongue, Early it is not painful but later become tender.
- ☒ **Black hairy tongue**: it is due to proliferation of chromogenic microorganisms causing brown staining of elongated filiform papillae.
- ☒ **Tumors of the tongue**: Squamous cell carcinoma & kaposi sarcoma.

Diseases of the salivary Glands

- ☒ **Ptyalism (Excessive salivation)**:
 1. Psychogenic & prior to vomiting
 2. Secondary to oral pathology e.g. stomatitis.
- ☒ **Xerostomia (dryness of the mouth)**:
eg. dehydration & sjogron .
- ☒ **Sialadenitis (inflammation of salivary glands)**: viral (mumps) & bacteria.
- ☒ **Salivary calculi**: occasionally in the submandibular gland or its ducts.

Eosophagus

Anatomy:-

- Muscular tube about 25 cm.
- closed proximally by the upper esophageal sphincter (UES)
- distally by the lower esophageal sphincter (LES)

Function:

- propel food or fluid into the stomach.
- Prevent reflux of gastric contents: between swallows the esophageal body is relaxed and collapsed while both sphincters are contracted.

Histology:

- Proximal 1/3 is striated muscles & distal 2/3 is smooth muscle fibers.
- The esophagus is lined by stratified Squamous epithelium except the gastro-oesophagageal junction is lined by columnar epithelium.

Dysphagia

Definition: Difficulty in swallowing due to (Mechanical or Motor) causes.

Etiology:

A. Mechanical causes:

I. Painful diseases of the mouth & pharynx: Stomatitis, pharyngitis, tonsillitis & retropharyngeal abscess.

II. Intrinsic disease of the oesophagus:

1. Congenital anomalies as atresia.
2. Gastro oesophageal reflux disease (GERD).
3. Stricture due to corrosive oesophagitis.
4. Tumors: Cancer oesophagus, sarcoma, lymphoma.
5. Plummer-Vinson syndrome.
6. Scleroderma.

III. Extrinsic Compression of the esophagus:

1. Cervical spondylosis
2. Thyroid enlargement
3. Left atrial enlargement
4. Zenker's diverticulum
5. Retropharyngeal abscess
6. Posterior mediastinal masses

B. Motor causes:

1. Neurological diseases

- Bulbar & pseudo-bulbar palsy.
- Neuritis & myasthenia gravis
- Polymyositis.

2. Functional: Achalasia & esophageal spasm.

Commonest causes of dysphagia: cancer - achalasia - post-corrosive

Approach to diagnosis of dysphagia:

☒ History:

1. Age: cancer in old age, post corrosive in children
2. Type of food: only to solids in mechanical causes, while to both solids and fluids in motor dysphagia.
3. Duration and course:
 - -short : inflammatory causes
 - Intermittent : Functional disorders
 - Progressive : cancer
4. Associated symptoms:
 - Severe loss of weight in cancer
 - Manifestations of thyroid dysfunction in cases with goiter
 - Nasal regurgitation in neurological causes.

☒ Physical examination:

1. **Neck:** for thyroid enlargement
2. **Features of scleroderma**

Investigations:

1. Endoscopy
2. Barium swallow
3. Esophageal manometry

2. Achalasia

Definition:

This is a motility disorder of the oesophagus where there is hypertonic lower oesophageal sphincter which fails to relax in response the esophageal swallowing wave, while the rest of the oesophagus shows progressive atony and dilatation.

Etiology:

The cause is unknown, with several theories involved:

- ✓ Failure of non-adrenergic non-cholinergic innervation
- ✓ Destruction of myenteric plexus
- ✓ Chaga's disease
- ✓ Degeneration of dorsal vagal nuclei within the brain stem in latter cases

Clinical Features:

1. The disease is more common in middle-aged females
2. Dysphagia to fluids and late to solids
3. Episodic chest pain that may resemble angina. (vigorous achalasia)
4. Purification of the retained residue leads to halitosis
5. Pulmonary infections may occur due to repeated aspiration.
6. There is progressive loss of weight.

Investigations:

1. Barium swallow:

- Dilated oesophagus with tapered pointed end
- Absence of the gas shadow in the fundus of the stomach.

2. Oesophagoscopy: Patients with achalasia have increased incidence oesophagus and esophagitis.

3. Esophageal manometry: to demonstrate high lower esophageal sphincter pressure

during swallowing, with poor peristalsis in the body.

Treatment:

1. Calcium blockers can be tried initially.
2. Pneumatic dilatation by a sdistensible bag, or injection of botulinum toxin.
3. Cardiomyotomy (heller's operation) if dilatation failed.

3. CarCinoma of the oesophagus

Risk factor

- ✓ Most cases (old men (60-70) year old heavy smoker or Alcoholic , spirits, celiac disease,
- ✓ tylosis (hyperkeratosis of palms and soles) & achalasia.
- ✓ Plummer-Vinson syndrome.

Pathology:

- **Macroscopically:** ulcerative - nodular - cauliflower mass
- **Microscopically:**
 - Squamous cell carcinoma (mostly lower 1/3)
 - Adenocarcinoma: in the lower third (Barret's esophagus)

Clinical Features:

- ✓ Progressive, painless **dysphagia**, first to solids, and later to fluids.
- ✓ Discomfort at the site of obstruction
- ✓ Weight loss
- ✓ Pulmonary aspiration
- ✓ Mediastinal syndrome, enlarged lymph nodes and liver affection may occur due to spread of the disease.

Investigations:

1. **Barium swallow: (filing defect +ve rat tail sign)**
2. **Oesophagoscopy :** For diagnosis & biopsy.
3. **CT chest and abdomen, endoscopic ultrasound:** for staging

Treatment:

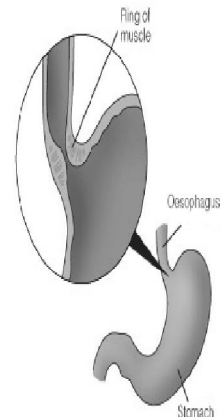
1. **Radical surqey**
2. **Palliative: Radio-therapy & stent.**



4. Gast ro-esophageal reflux disease (GERD)

Definition:

Reflux of gastric contents into the oesophagus which causes symptoms & may be complications.



Patho-physiology:

- Occasional episodes of Gastro-esophageal are common in healthy individuals.
- It is normally followed by esophageal peristaltic waves which efficiently clear the gullet
- Alkaline saliva which neutralizes acidity
- Gastro-esophageal reflux disease develops when oesophageal mucosa is exposed to gastric contents for prolonged period of time.

Aetiology = failure of anti reflux :(3 i & 3D)

1. **I**mp**ro**per relaxation of lower oesophageal sphincter (LES) while lying flat.
2. **I**n**cr**eased intra-abdominal pressure: pregnancy, tight clothes and obesity.
3. **H**i**at**us hernia may aggravate the condition.
4. **D**e**cr**eased acid clearance due to impaired oesophageal peristalsis.
5. **D**e**l**ayed gastric emptying
6. **D**i**e**tary and environmental factors: coffee, fat and chocolate relax the lower esophageal sphincter and may provoke symptoms.

Clinical Picture:**A. Oesophageal:**

1. Heart burn: the most common symptom, associated with water brash.
2. Chest pain: simulating angina due to reflex esophageal spasm.
3. Odynophagia: painful swallowing
4. Dysphagia: due to disturbed motility or structure.

B. Extra-oesophageal:

1. Pulmonary: cough, asthma & aspiration pneumonia
2. Oropharyngeal & laryngeal irritation
3. Iron deficiency anemia (esophagitis)
4. Atypical chest pain

Complications: (ABBE)

1. Esophagitis: The severity of the endoscopic findings does not correlate with symptoms.
2. Barret's: columnar lined esophagus with area of intestinal metaplasia occur as an adaptive mechanism for chronic GORD. It is found in 10% of cases. It is premalignant with cancer risk 0.5% year.
3. *Anemia*: due to chronic blood from Esophagitis.
4. Benign esophageal stricture.

Investigations:

1. **Endoscopy:** to diagnose complications e.g. oesophagitis. Biopsies from areas of Barrett's esophagus every 2-3 years if there is no metaplasia or every 12 months in low grade dysplasia.
2. Esophageal PH monitoring
3. Esophageal manometry
4. Esophageal Barium study: may detect hiatus hernia.

Treatment:**A- General measures:**

1. Avoid alcohol, smoking, fatty meals and nitrates.
2. Avoid large meals especially before sleep
3. Weight reduction.
4. Elevation of the head of the bed

B- Drug therapy:**1. Drugs Decreasing gastric acidity:**

- ✓ *Antacids & H₂ blockers:* (Cimetidine, ranitidine or famotidine). For mild cases. They relieve symptoms but don't heal esophagitis.
- ✓ *Proton pump inhibitors:* (Omeprazole, Lansoprazole or pantoprazole)

2. Drugs Regulating motility:

- ✓ Domperidone (Motilium)
- ✓ Metoclopramide (Primpiran)

C- Surgery: For cases with failed medical treatment, Nissen fundoplication

5. Other esophageal disorder

1- Diffuse esophageal spasm: Severe form of abnormal esophageal motility

2- Globus (globus hystericus): Persistent or intermittent sensation of lump or foreign body in the throat between meals - No dysphagia

Treatment: reassurance

3- Esophageal perforation

Etiology: 1- Trauma e.g. endoscopy

2- Sudden increase in intra esophageal pressure e.g. violent vomiting (Boerhaave's syndrome)

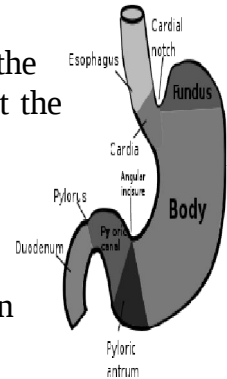
C/P:- Chest or abdominal pain - Shock

Treatment: IV fluids and antibiotics - Surgery in severe cases

Stomach and duodenum

Physiological aspects:

- HCL is secreted by the parietal cells of the stomach due to the activity of the hydrogen-potassium ATPase (proton pump), at the parietal cell membrane.
- The parietal cell has 3 receptors for its stimulation.
 - M3 receptor: stimulated by acetylcholine.
 - H2 receptor: stimulated by histamine,
 - G receptor: stimulated by gastrin, derived from G cells in the antrum
- Somatostatin derived from D cells inhibits acid secretion.
- Pepsinogen is secreted by the chief cells of the stomach. Acid converts pepsinogen to pepsin.
- Bicarbonate ions & mucus protect the gastroduodenal mucosa from the ulcerative properties of acid & pepsin.



1-Peptic ulcer disease (Pud)

Definition:

An ulcer is a break in the mucosa occurring due to injury by acid & pepsin, in the duodenum stomach, lower oesophagus or in the jejunum after surgical anastomosis to the stomach.

Pathology:

- ▲ Ulcers extend through the muscularis mucosa and are usually 5 mm in diameter.
- ▲ Duodenal ulcers are 5 times more common than gastric ulcers.
- ▲ 95% of the duodenal ulcers are in the bulb or pyloric channel.

Aetiology:

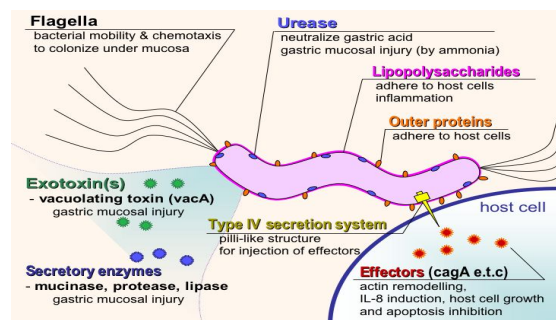
1. Helicobacter pylori (HP):

☒ **Organism:** spiral gram-negative rod beneath the gastric mucosal layer

☒ **Mode of transmission:** From person to person by gastro-oral root.

☒ **Epidemiology:**

- The prevalence of H.pylori infection among general population is increasing with age. For adults, the prevalence is 50% in the developed and up to 90% in the underdeveloped countries.
- There is accumulating evidence that H. pylori is the main factor for causing peptic ulcer in people not receiving NSAIDs. And the main factor for causing recurrence after treatment.



☒ Pathogenesis:

- ✓ The organism lives in the mucus adherent to epithelium.
- ✓ The organism may produce a transient acute gastritis with PMNLS infiltration
- ✓ It produce some toxic products which leads to gastritis , ulcer as in figure(1) .:

2. Non-steroidal anti-inflammatory drugs (NSAIDs):

- ✓ Prostaglandins play a major role in gastric protection as they stimulate bicarbonate and mucus secretion and increase mucosal blood flow.
- ✓ NSAIDs (anti-inflammatory agents) which act by inhibiting cyclo-oxygenase enzyme (COX) activity, which decreases prostaglandins at the site of inflammation.
- ✓ It is estimated that 30% of peptic ulcers are caused by NSAIDs.
- ✓ Recently it was found that cyclo-oxygenase enzyme (COX) has 2 types COX-2 which is present at sites of inflammation and COX-1 which is present in the gastrointestinal mucosa.
- ✓ Selective COX-2 inhibitors, which decrease inflammation and do not injure the gastrointestinal mucosa are available now.

3. Smoking:

Increases ulcer complications, decreases ulcer healing with standard therapy, and increases the rate of ulcer recurrence.

4. Heredity:

- ✓ Family history may be positive in some cases.
- ✓ PUD is more common with blood group "O"

5. Other factors:

- ✓ GERD is responsible for ulcers at distal oesophagus.
- ✓ Gastrinoma, is responsible for P.U. in Zollinger-Ellison syndrome.
- ✓ P.U. is more common in cirrhotics due to diminished destruction of gastrin & histamine by the liver and due to diminished mucosal resistance due to congestion caused by portal hypertension.
- ✓ There is no strong evidence for a relation between psychological factors or diet with peptic ulcer disease.
- ✓ P.U. is more common in men than women.

Clinical Picture:

Symptoms:

1. Pain:

- ✓ Character: Burning, stabbing, or dull.
- ✓ Site:
 - ▶ D.U.: Above the umbilicus & to the right.
 - ▶ G.U.: Epigastric & in the mid line.
 - ▶ Penetrating ulcer – Pain in the back
- ✓ Relation to meals :
 - ▶ D.U. : 2-4 h after meals & at night.
 - ▶ G.U.: 0.5-1 h after meals.
- ✓ Nocturnal pain: Pain awaking the patient is highly diagnostic of D.U.

✓ Relief of pain:

- D.U.: Food & alkalis, but not vomiting.
- G.U.: Vomiting & alkalis, but not food.

✓ Chronicity & periodicity: this is a characteristic feature, especially in D.U.

2. Vomiting:

- Usually present in G.U. but uncommon in D.U.
- Significant vomiting or weight loss suggests gastric outlet obstruction or malignancy

3. Heart burn & water brush : in GERD.

4. Appetite: is normal, but: in D.U., the patient eats frequently to relieve pain. In G.U., the patient afraid to eat for fear of pain.

5. The patient may present with complications:

- a) G.I. bleeding: Haematemesis or melaena (Melaena is commoner in D.U., haematemesis, is commoner in G.U.), occult blood in stool.
- b) Perforation : It usually presents with sudden onset of severe abdominal pain, ileus and board like rigidity of the abdomen. The symptoms may be less severe in old age or in patients on NSAIDs and the first presentation may be shock or septicemia.
- c) Penetration: into the pancreas or the biliary system presents with increasing severity of pain with radiation to the back and symptoms and signs of pancreatitis or cholangitis.
- d) Chronic pyloric obstruction or hour glass stomach:
 - The obstruction may be due to oedema or fibrosis around the ulcer
 - It presents with epigastric fullness, vomiting of foul odorous previously ingested food, with alkalosis (possibly tetany) and loss of weight in long standing cases.

Signs:

1. May be absent
2. Localized tenderness over the site of the ulcer.
3. Signs of complications if present e.g. succussion splash in pyloric obstruction.

Investigations:

A. Endoscopy:

This is the best method to detect an ulcer and to differentiate between benign & malignant ulcers by biopsy.

B. Barium meal: (rarely used now)

- a) In gastric ulcer: Persistent ulcer niche on the lesser curvature with notch on the greater curvature opposite the niche.
- b) In duodenal ulcer: Persistent deformity of the duodenal cap.

C. Investigations for helicobacter pylori:

1. Serum antibodies: Detected by ELISA & Positive tests do not indicate active infection as antibodies level declines after eradication of the organism in 50% of patients by 12-18 months.
2. Fecal antigen immunoassay: a positive test indicates active infection.
3. Histological identification: in endoscopic biopsies.
4. Urease test:
Biopsies are added immediately to a solution of urea, if HP is present the urea is broken down by the urease to produce a colour change in the indicator, in a short time.
5. Culture & sensitivity: from endoscopic biopsies, takes 3-7 days.
6. The 13C-urea and 14C-urea breath test: it indicate active infection, however, their greater cost make them less utilized in most clinical settings.

Differential Diagnosis:

1. From other causes of upper abdominal pain.
2. From other causes of dyspepsia
3. From other causes of G.I. bleeding.

Treatment:**A. General measures:**

- Stop NSAIDs.
- Balanced meals at regular intervals
- Stop smoking

B. Medical Treatment:**1. H₂ blockers:**

Peptic ulcer will heal after 2 months course

Several drugs are available:

- Cimetidine (Tagamet): 800 mg/D (SE: gynecomastia&impotence)
- Ranitidine (Zantak): 300 mg/D.
- Famotidine (Gastrodmina): 20 mg/D.

2. Proton Pump Inhibitors (PPIs);

- They permanently inhibit H-K ATPase.
- These produce a 90% inhibition of gastric acidity (compared to 65% in H₂-blockers) & healing rate of 90% after 4 weeks treatment (for DU) and 8 weeks (for GU).
 - Omeprazole (Losec): 20 mg/D
 - Lanzoprazole (Lanzor): 30 mg/D
 - Pantoprazole (Controloc): 20 mg/D

They should be administered 30 minutes before breakfast.

3. Agents enhancing mucosal defences:

Sucralfate:

Structure: Aluminum hydroxide and sucrose sulphate, 1gm TDS before meals.

Mechanism: It inhibits H⁺ diffusion to ulcer base, and promotes mucus bicarbonate, and endogenous PGs to help healing.

Side effects: neurotoxicity of aluminum in renal failure patients.

Bismuth containing agents: stimulate PGs secretion and effective against H. pylori

Prostaglandin E1 analogue (Misoprostol):

Mechanism: increases mucosal blood flow, mucous and bicarbonate secretion and repair.

Dose: 200 ug tab TDS

Side effects: diarrhea and abdominal pain

Given the efficacy and safety of antisecretory agents, these agents are no longer used as first line therapy for active ulcers.

4. Anticholinergic agents:

- **Drug:** Pirenzepine (Gastrozepine) selective M1 blocker
- **Action:** ↓ gastric secretion and delays gastric emptying
- **Side effects:** dryness of mouth, tachycardia

5. Eradication of H. Pylori:

- Several regimens are used
- The most common is Triple therapy for 2 weeks.
- Metronidazole (400 mg TID) + amoxicillin (750 mg TID) or clarithromycin (500 mg TID) + proton pump inhibitor.
- Hel-cure: (Tinidazole + omeprazole + clarithromycin) tab/12 hrs for 2 weeks.

C. Endoscopic therapy:

- Used in treatment of bleeding ulcer by injection of adrenalin or clipping.

D. Surgical Treatment: indicated in

1. Uncontrollable hemorrhage. Under medical treatment.
2. Perforation
3. Pyloric obstruction
4. Malignancy

E. Treatment of Complications:

1. Haemorrhage:

- Omeprazole: by I.V infusion.

- ▶ Ranitidine: by I.M injection
- ▶ Endoscopic injection of adrenaline or clipping
- ▶ Blood transfusion: if needed
- ▶ Surgery: if other measures fail

2. Perforation : I.V. fluids, analgesics, antibiotics then surgery

3. Pyloric obstruction: Gastric lavage, I.V. fluids then surgery

4. Malignancy: Surgical removal.

2- Acute gastritis

Definition: Inflammation of the superficial gastric mucosa

Etiology:

- ♦ **DRUGS**: Aspirin, NSAIDs.
- ♦ **STRESS**: (Stress gastritis) : Sepsis, Shock, Severe burns (Curling's ulcer), cerebral trauma and stroke (Cushing ulcer)
- ♦ **INFECTIONS**: CMV, HSV.

Pathology:

- ♦ Mucosal inflammation occurs due to increased acid production along with decreased mucosal blood flow and surface bicarbonate.
- ♦ Mucosal infiltration with neutrophils and sloughing occurs.

Clinical picture : Epigastric pain, nausea, vomiting and hematemesis

Investigations: Upper endoscopy confirms the diagnosis

Treatment:

- ♦ Removal of the offending cause.
- ♦ **Drugs**: H₂ receptor antagonists (ranitidine 150 mg bid). Proton pump inhibitors (omeprazole 20 mg once daily)
- ♦ Gastric wash by cold saline and gastrectomy in severe cases

3- Chronic gastritis

Definition: it is chronic inflammation of gastric mucosa which can lead to mucosal atrophy.

Types:

1. Autoimmune gastritis (**Type A**): it occurs in **Pernicious anemia**.
2. Chronic active gastritis (**type B**): due to infection by *Helicobacter pylori*.
3. Chemical gastritis (**Type C**): due to repeated chemical injury, e.g. bile reflux
4. Chronic NSAIDs ingestion

c/p: asymptomatic, may be epigastric pain & nausea - vomiting

Treatment: Treatment of the cause: e.g. *H. pylori*, pernicious anemia.

4- Functional gastro-duodenal disorders

A. Gastroparesis

Etiology:

1. Autonomic neuropathy e.g. DM, uremia
2. Scleroderma
3. post gastric surgery

C/P: nausea, vomiting & early satiety.

- ✓ Treatment: Prokinetic agents e.g. domperidone (motilium)
- ✓ Erythromycin: stimulates GIT motilin receptors

B. Non ulcer dyspepsia (functional dyspepsia): look dyspepsia

Gall Bladder and Biliar Gall Bladder and Biliary SyStem

1-Gall bladder stones

Aetiology:

A. Cholesterol stones: 8%

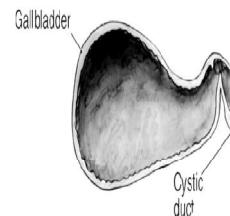
- Cholesterol stones develop when the liver produces bile with excess cholesterol or low bile salts.

B. Pigment stones: 20%

1. Black pigment stones: due to chronic haemolysis.

2. Brown pigment stones: due to

- Stagnation of bile in common bile duct e.g. by stricture.
- Infection: bacterial β -Glucuronidase splits conjugated bilirubin to its free form precipitates as calcium bilirubinate.



Clinical Picture: Usually (5F) female, fatty, fertile, fair, forty:

1. Asymptomatic (90% of cases)
2. Pain: Pain occurs suddenly and persists for 2 hours (if it persists more than 6 hrs this indicates complications), Pain is usually felt in the epigastrium (70% of cases) or the right hypochondrium (20%) and radiates to the interscapular region or the tip of right scapula.
3. Obstruction of cystic duct: biliary pain or cholecystitis.
4. Obstruction of common bile duct: pain, jaundice, cholangitis.

Investigations:

1. X-ray: demonstrate calcified gall stones (20% of cases)
2. Abdominal ultrasonography, CT scan and MRCP

Complications:

1. Mucocele or pyocele
2. Porcelain gall bladder
3. Choledocholithiasis
4. Fistula between gall bladder and duodenum, colon or stomach
5. Gall stone ileus
6. In 95% of gallbladder cancer there are accompanying gallbladder stones

Treatment:

1. Asymptomatic cases: no treatment
2. Medical dissolution of the stone:

Indications: non-calcified stones, smaller than 15mm, mild symptoms and moderate obesity

Drug: chenodeoxycholic acid (12 mg/kg/day) or ursodeoxycholic acid (12 mg/kg/day)

3. Extracorporeal shock wave lithotripsy.
4. Surgical or laparoscopic cholecystectomy
5. Treatment of complication

2- Acute cholecystitis

Aetiology:

A. Obstruction of the cystic duct by stones: (96%)

- The retained bile salts cause sterile inflammation.
- G.B. may distend with mucus (mucocoele) or pus (empyema).
- Increased pressure compresses blood vessels, causes gangrene.

B. Bacterial inflammation :

- Common organisms are E. coli & strept, faecalis.
- The bacteria deconjugate bile salts into irritant bile acids. Culture of bile detects.
- Organisms in 50% of the cases.

Clinical Picture:

1. Severe pain usually in the right hypochondrium or epigastrium. It can radiate to the back and to the right shoulder. Pain lasts for hours.
2. Tenderness and rigidity in the right hypochondrium which is worse on inspiration (**Murphy's sign**)
3. Gallbladder mass may be felt
4. Flatulence, nausea & vomiting may occur.
5. Fever (rigors unusual), jaundice (10% of cases with passage of stones to CBD)

Investigations:

Blood: Leucocytosis, AST, ALT, bilirubin & alkaline phosphatase: may be elevated.

Ultrasound : G.B tenderness & thickening, may be distension.

Radionuclide HIDA scan.

Differential Diagnosis:

- | | |
|-------------------------------|-----------------------------|
| 1. Acute appendicitis | 2. Diaphragmatic pleurisy |
| 3. Acute pancreatitis | 4. Muscular or root lesions |
| 5. Acute right pyelonephritis | 6. Myocardial infarction |
| 7. Perforated peptic ulcer | |

Prognosis:

1. Spontaneous recovery in 85% of patients, Recurrent attacks of acute cholecystitis may follow.
2. Rarely, gangrene or empyema of the gall bladder, fistula formation, hepatic abscess or even generalized peritonitis may occur.

Treatment:

1. Medical treatment:

- Bed rest, I.V. fluids, light diet.
- Relief of pain by pethidine
- Antiemetic as domperidon suppositories
- Antibiotics: third generation cephalosporin or a quinolone + Metronidazole is usually given in combination.

2. Surgery:

- Emergency surgery: with complications or failed medical treatment.
- Elective surgery: better done after three days of medical therapy.

3- Chronic Calcular Cholecystitis

Clinical Features:

- ✓ It is usually contracted with thickened wall.
- ✓ stones is almost constant association.
- ✓ There are recurrent attacks of right upper abdominal pain
- ✓ tenderness over the gall bladder and a positive Murphy's sign .

Investigations: Ultrasonography shows gall bladder stones & thickened gall bladder

Treatment: cholecystectomy.

4- other Disorders

Post cholecystectomy syndrome

- ✓ Rt hypochondrial pain, flatulence, intolerance fatty meals despite normal image of biliary tract.
- ✓ Most of cases have functional bowel disorder and were diagnosed falsely as cholecystitis because of incidental GB stone in sonar.

Primary sclerosing cholangitis

- Autoimmune inflammation and fibrosis of intra and extra hepatic bile ducts.
- Ulcerative colitis is a common association (70% of cases)
- Asymptomatic or Obstructive jaundice and liver cirrhosis.
- ANCA antibodies in 80% of cases
- treatment:- Steroids, azathioprine, ursodeoxycholic acid.

Acute cholangitis

Bacterial infection of bile ducts due to obstruction or following ERCP

C/P: (charcoat's triad) **Fever &rigors, jaundice**

Investigations: Leucocytosis, positive blood culture (usually E. coli)

Treatment: Antibiotics (Ceftazidime + gentamycin) + treatment of obstruction

5- Miscellaneous biliary disorders

- ☒ **Hemobilia:** Bleeding in common bile duct
 - Etiology : trauma, tumor, or, or liver biopsy.
- ☒ **Biliary dyskinesia:** Recurrent right upper quadrant and Epigastric pain
 - Etiology : increased tone of sphincter of oddi.
 - Treatment: nitrates, Ca channel blockers or ERCP sphincterotomy.
- ☒ **Caroli's syndrome:** congenital segmental dilatation of intrahepatic biliary system.

PANCREAS

1- Acute pancreatitis

Def: Acute inflammation of the pancreas due to autodigestion by prematurely activated pancreatic enzymes.

Aetiology:

- 1- **Gall stones** (commonest)
- 2- Alcohol
- 3- Infections e.g. mumps,
- 4- Drugs e.g. azathioprine, thiazide.
- 5- Pancreatic tumours
- 6- Iatrogenic e.g. ERCP, post surgical
- 7- Metabolic: hyperlipidaemia, hypercalcemia, severe hypothermia.
- 8- Idiopathic

Pathophysiology:

- premature activation of proteolytic enzymes in the pancreas,
- Which will digest the pancreas and the surrounding tissues.
- The severity depends on the balance between the proteolytic enzymes and the antiproteolytic factors (α_1 anti-trypsin, β_2 macroglobulin and C1 esterase inhibitors)

Clinical Picture:

1. Epigastric abdominal pain which may radiate to the back
2. Nausea & vomiting.
3. Epigastric tenderness however, rigidity and rebound tenderness may be absent as the inflammation is retroperitoneal.
4. Rarely, abdominal wall ecchymosis e.g.:
 - ▲ Cullen's sign : umbilical ecchymosis.
 - ▲ Grey Turner's sign : ecchymosis in the flanks.
5. Features of complications.

Complications:**1. Pancreatic:**

- ♦ Phlegmon (inflammatory mass)
- ♦ Pseudocyst: it consist of tissue debris, with enzyme rich pancreatic fluid
- ♦ Pancreatic abscess
- ♦ Ascites

2. Intestinal: Paralytic ileus & G.I. haemorrhage.**3. Hepatobiliary:** Obstructive jaundice & portal vein thrombosis.**4. Metabolic:** Hypocalcaemia & hypoglycaemia.**5. Haematological :** Disseminated intravascular coagulopathy.**6. Renal:** Acute renal failure.**7. Cardiovascular:** Circulatory failure (shock).**8. Respiratory:** ARDS.Investigations:**1. Serum amylase:**

- Raised (5 time greater than normal). Returns to normal 2-3 days (effectively excreted in the kidney, & in these cases there is increased urinary amylase : creatinine ratio)
- Persistently elevated amylase indicates pseudocyst.
- It may elevated in e.g. intestinal ischemia, perforated ulcer

2. Other laboratory tests:

- WBC count: usually increases to 12000-20000/ml
- Hyperglycemia
- Hypocalcaemia: D.T. pancreatic calcification & hypoalbumemia.
- Increased serum bilirubin (15-25%): compression of bile duct

3. Plain X-ray abdomen: Ileus, calcified gall stones & panc. calcification**4. Ultrasound:** swollen pancreas, fluid collections, gallstones.**5. Contrast enhanced CT:**

- ♦ Visualization of pancreatic viability
- ♦ The presence of gas among necrotic tissue indicates infection

6. MRI & MRCP: degree of pancreatic damage other involvement**7. Peritoneal aspiration & lavage : contains high amylase.**Treatment:**1. Nasogastric suction, nothing by mouth & give parenteral nutrition.****2. Analgesia:pethidine (50-100mg /4 hrs) avoid morphine as it may cause spasm of sphincter of Oddi.****3. ↓ Pancreatic secretion by somatostatin, glucagon and anticholinergic infusion have no proven benefit.****4. ERCP to remove the bile duct stones**

5. **P**rophylactic antibiotics (imipenem IV 500 mg TDS)
6. **P**latelet activating factor inhibitor (Lexipafant)
7. **P**eritoneal lavage is of doubtful value.
8. Management of complications

Prognosis: (within the 1st 48 hrs). (ABCDE)

1. **Age**: old (more than 55 years)
2. **Albumin** : diminished (less than 3 gm /100 ml)
3. **Arterial O₂**: diminished (less than 60 mmHg)
4. **Blood glucose**: raised (more than 200 mg/100ml)
5. **Blood urea**: raised (more than 100 mg/100 ml)
6. **WBC's**: raised (more than 15,000/mm³)
7. **Decrease Serum calcium** : diminished (less than 8 mg%)
8. **Elevated Serum transferases**: raised (more than 200 units)

<3 = mortality 1% - 3-5 = mortality 10-20% - >6 = mortality >50%

2- Chronic pancreatitis

Aetiology:

1. Chronic alcoholism (85% of cases): the most common cause
2. Tropical, hereditary : the most common cause in developing countries.
3. Obstructive e.g. pancreatic duct stricture..

Clinical Picture: A triad of

- i Abdominal pain resembling cases
- i Steatorrhoea
- i Diabetes

replacement therapy & ttt of DM

3- Carcinoma of pancreas

Predisposing factors:

- **Smoking & fat & chronic pancreatitis & petroleum products**

Pathology:

- 90% adenocarcinomas arise from the duct epithelium. & 70% arising from the head of pancreas.

Clinical picture:

It affects males more than females above age of 60.

1. **Carcinoma of the head** Painless jaundice & wt loss & pain
2. **Carcinoma of the body or the tail**: pain & rare Jaundice & wt loss & Glucose intolerance may occur.

3. Courvoisier's sign:

Dilatation of gall bladder may occur with cancer head pancreas, this is usually not found with gall stone disease due to the associated chronic inflammation with fibrosis of the wall of gall bladder.

Investigations:

- Tumor markers as CEA (carcinoembryonic antigen)
- Spiral contrast enhanced CT.

Treatment:

- Whipple operation (removal of the duodenum and the head of the pancreas)
- palliative.

DISEASES OF INTESTINE

1- Malabsorption syndrome

Definition:- Defective absorption of any or all of the food components absorbed from the intestine. The main defect in absorption of fats leading to steatorrhea "fatty stools".

Etiology:

A. Gastric causes:

- ✓ Gastric surgery:- e.g. gastrectomy.
- ✓ Gastric carcinoma:- decreased HCL leading to bacterial overgrowth in the intestine.
- ✓ Gastrinoma:- increased HCL leading to inhibition of digestive enzymes.

B. Pancreatic causes:

- ✓ Chronic pancreatitis.
- ✓ Cystic fibrosis of the pancreas
- ✓ Pancreatic carcinoma.
- ✓ Pancreatectomy

C. Hepato-biliary:

- ✓ Hepatic causes: Acute & chronic hepatitis.
- ✓ Biliary causes: Obstructive jaundice.

D. SMALL INTESTINAL DISEASES: (1st & 2nd ry)

☒ **Primary:-** celiac disease & tropical sprue

☒ **Secondary:-** (3S+3I+---)

1. Stagnant (blind) loop syndrome

Strictures, surgery (Roux en Y operation), Diverticulosis, Diminished motility (e.g. DM, scleroderma) lead to stagnation of intestinal contents with overgrowth of bacteria with diarrhea and malabsorption.

2. **Short bowel syndrome** : excessive intestinal resection will reduce the available surface for absorption.
3. **Systemic disease as Amyloidosis**
4. **infections:-** TB - Giardia ----
5. **irradiation enteritis:-**
6. **inflammation:-** inflammatory bowel disorder
7. **Disaccharidase deficiency:-** Lactase deficiency = malabsorption of lactose bloating & diarrhea.
8. **Lymphatic obstruction:- Lymphoma & whipple's disease**

Whipple's disease:- flattening of the intestinal villi which are heavily infiltrated by macrophages with obstruction of intestinal lymphatics.
C/p:- Malabsorption, arthritis, lymphadenopathy & cranial nerves affection.
Investigations:- intestinal biopsy, PCR
Treatment:- penicillin, tetracycline, or sulphonamides.

E. DRUG –INDUCED:

- Cholestyramine
- Neomycin

F. MISCELLANEOUS:-

1. Heart : Congestive heart failure & Constrictive pericarditis.
2. Thyroid: Myxoedema
3. Adrenal insufficiency

Celiac disease: "Gluten – sensitive enteropathy"

Etiology and pathogenesis :Immunologically mediated inflammatory disorder of the small intestine occurring in genetically susceptible individuals due to reaction against gliadin (one of gluten peptides) leads to activation of lymphocytes in the lamina propria of the intestine with the release of cytokines that leads to the atrophy of intestinal villi and crypts hyperplasia. There is high association with HLA-Dq2.

- Clinical picture: - Age:3rd-4th decade.
 - Females are more affected than males
 - Full C/P of Malabsorption syndrome or non-specific symptoms as diarrhea or anemia.

Investigations: Serum level of antigliadin antibodies - anti-endomysial antibodies (IgA) + investigation of malabsorption

Treatment: Gluten free diet - steroids

Tropical sprue

Etiology: may be bacterial infection.

Clinical picture: Severe malabsorption, especially of fats, folic acid & cobalamin.

Investigations: Jejuna biopsy: may show villous atrophy + investigations for malabsorption.

Treatment: Antibiotic : Tetracycline 250 mg /6 hours orally daily.

Replacement: Folic acid & cobalamin

Clinical Picture:**A. General features of malabsorption:**

- Steatorrhea: stools are bulky, greasy, offensive, and difficult to flush.
- Diarrhea.
- Abdominal distension, colics, borborygmi (audible intestinal sounds).

B. Deficiency of specific factors:

- Fats: Loss of weight.
- Proteins: Muscle wasting & nutritional oedema.
- Carbohydrates: hypoglycemia.
- Vitamin A : Night blindness.
- Vitamin D : Rickets or osteomalacia
- Vitamin E : Dermatitis.
- Vitamin K : Bleeding tendency
- Vitamin B1 : Beri-Beri.
- Vitamin B2 : Glossitis & angular stomatitis.
- Nicotinic acid : Pellagra
- Vitamin B6 : Peripheral neuropathy.
- Vitamin B12: Megaloblastic anemia & SCD.
- Folic acid : Megaloblastic anemia.
- Iron: Microcytic anemia.
- Sodium : Muscle cramps & hypotension
- Potassium : Myopathy & arrhythmia
- Calcium : Parasthesia & tetany
- Magnesium: arrhythmia & tetany.
- Water : Dehydration.

C. Features of the cause**Investigations:-****A. Investigations to diagnose malabsorption:****1. Fat in the stools:**

- Normally : fat in the stools is not more than 6 gm/day
- In steatorrhea: total fat is increased:
- Non-split: in impaired digestion (pancreatic disorder)
- Split: in small intestinal diseases.

2. Urinary D-xylose test :-

- Method: give 25 gm D- Xylose orally, then collect urine over the next 5 hours.
- Normally: urine collected over 5 hours should contain at least 5 gm of D – Xylose.
- In steatorrhea: urine collected over 5 hours will contain less than 5 gm of D – Xylose " provided that renal functions are normal".

3. Barium follow through of the small intestine:

- Loss of the normal feathery appearance of the jejunum.
- Segmentation & flocculation of the barium pattern.
- Dilatation of the intestinal lumen.

4. Jejunal Biopsy:

- Recently : taken through the enteroscope.
- Previously: taken by the intestinal biopsy capsule (Crosby capsule).

B. Investigations to diagnose the cause:**1. Glucose tolerance test:**

- In small intestinal diseases: flat curve.
- In pancreatic causes : diabetic curve.

2. Tests for Bacterial overgrowth:

- 14 C-glycocholic breath test:
 - Method: measure the 14 CO₂ in the breath after oral administration of 14 C- glycocholic.
 - In bacterial overgrowth : 14 CO₂ in the breath will increase because more bacteria will act on the 14 C – Xylose.
- Culture of the jejunal aspirate:
 - This is done using a sterile polyethylene tube.
 - Normally there is <10000 organisms/ml.

C. Investigations for the deficient factors:

1. Blood picture : microcytic or megaloblastic anemia
2. Plasma proteins: hypoproteinemia
3. Serum electrolytes: diminished iron, Na, K, Ca, Mg.
4. Bone X –ray: osteomalacia.

Treatment:

1. Treatment of the cause: e.g. anti – TB drugs for TB.
2. Replacement of the deficient factors : e.g. parenteral vitamins & minerals.

2-Dysentery

Definition :

A disease characterized by : frequent watery stools, blood & mucus in the stools + tenesmus.

Causes: ABCDE

1. Amoebic dysentery
2. Bacillary dysentery.
3. **B**ilharzial dysentery.

4. Cancer colon OR cancer rectum.
5. Crohn's disease of the colon OR ulcerative colitis.
6. Diverticulosis
7. Mercury poisoning
8. Uremic dysentery.
9. Protozoa : Malaria OR Giardia Lamblia

1-Amoebic dysentery

Etiology:

- **Organism :** Entamoeba Histolytica
- **Transmission:** by fecal – oral route.
- **Mode of infection:**
Ingestion of the cyst which resists gastric acidity & is transformed into "vegetative form" in the intestine & then passes to the colon.
- **Source:** Patient or carrier (cyst passer).

Clinical Picture:

A. ASYMPTOMATIC CYST PASSERS:

They pass cysts in the stools & cause spread of infection.

B. ACUTE AMOEBIASIS:

1. ACUTE AMOEBIC DYSENTERY:

- ♦ Acute diarrhea: Frequent motions, may be 10 motions per day.
- ♦ Associated symptoms: abdominal colicks, tenesmus, tenderness over the caecum & sigmoid.
- ♦ Stools: bulky, with mucus, blood & may be pus in secondary infection
- ♦ General condition : No fever, toxemia or dehydration.
- ♦ Course: the condition subsides in few days or turns into chronic form.

2. ACUTE AMOEBIC DIARRHEA:

C. CHRONIC AMOEBIASIS:

1. Chronic diarrhea : recurrent attacks of diarrhea.
2. Chronic dysentery.
3. Constipation
4. Abdominal pain:
5. Over the caecum or sigmoid colon.

Complications:

1. Amoebic hepatitis : & Amoebic liver abscess.
2. Amoeboma: mass in the right iliac fossa.
3. Amoebic ulcers: which may rarely perforate.
4. GIT bleeding

Investigations:

1. **Serology:** Amoebic antibodies may be detected in the serum of the patient.
2. **Stool analysis:** May show *Entamoeba Histolytica*: vegetative form OR cyst form.
3. **Sigmoidoscopy:**
May show amoebic ulcers (flask – shaped ulcers with healthy mucosa in between) with positive swab for amoeba.

Differential Diagnosis:

- From other causes of acute diarrhea.
- From other causes of chronic diarrhea.
- From other causes of dysentery.

Treatment:***A. Specific treatment:***

1. **Luminal amoebicidals:**
 - Indication: for treatment of cyst passers.
 - Drugs: Furamid: 500 mg TDS orally for 5 days.
2. **Tissue amoebicidals:**
 - Indication : for treatment of acute intestinal & extra – intestinal amoebiasis
 - Drugs:
 - a. Emetine hydrochloride (refer to amoebic liver abscess).
 - b. Dehydro-emetine
 - c. Chloroquine: effective ONLY in amoebic liver abscess.
3. **Tissue & Luminal amoebicidals:**
 - Indication: for acute intestinal amoebiasis, & amoebic liver abscess.
 - Drugs:
 - a. Metronidazole (Flagyl) : 750 mg tds orally for 10 days.
 - b. Tinidazole (Fasigyn) : 2 gm daily orally for 5 days

B. Symptomatic treatment: Antispasmodic drugs, Antidiarrhoeal drugs.**2-Bacillary dysentery****Etiology:**

- **Organism:** Gram – ve: *Shigella*, *S. dysenteriae*, *S. flexneri*, *S. boydii*, *S. sonnei*.
- **Mode of infection:** Fecal – Oral
- **Source :** the infection is commonly transmitted by flies from person to person.

Clinical Picture:

1. **GIT manifestations:**
 - Acute diarrhea: watery with frequent motions, may be 15 motions per day.

- Associated symptoms : abdominal colics, tenesmus, tenderness over the caecum & sigmoid.
- Stools : scanty, with excessive pus, blood & mucus.

2. General manifestations: fever, toxmia, dehydration.

Complications:

1. Bacteremia
2. Osteomyelitis & arthritis
3. Iridocyclitis & conjunctivitis.
4. UTI

Investigations:

1. Stool analysis:

- Excessive WBCs & RBCs.
- Culture may demonstrate the organism.

2. Sigmoidoscopy:

- Generalized inflammation of the colon.
- Dirty yellowish pseudomembrane which bleeds when removed.

Treatment:

A. Symptomatic treatment:

1. Antispasmodic drugs: for severe abdominal colics.
2. Correction of dehydration: Ecess oral fluids & IV fluids in severe conditions.

N.B. Antidiarrhoeal drugs: SHOULD NOT BE GIVEN because they may decrease the intestinal motility and thus decrease the clearance of bacteria, leading to more deterioration.

B. Specific treatment: Ciprofloxacin 500 mg bid orally for 5 days.

3-Bilharzial dysentery

Etiology:

- It is due to S. Mansoni ova which live in the mesenteric plexus of veins.
- It is very rarely due to S. Haematobium which live in the vesical plexus of veins due to vesicomesenteric anastomosis.

Clinical Picture:

1. Asymptomatic : most of the cases.

2. Intestinal manifestations: "Bilharzial dysentery"

- It occurs in cases of polyps or ulcers in the colon.

- It presents mainly with bleeding per rectum but it may present by chronic diarrhea & dysentery.
- It may be associated with a pericolic mass in the left iliac fossa.

3. Extra – intestinal manifestations:

- Anemia of chronic disease.
- Clubbing
- Hepatic Schistosomiasis
- Bilharzial cor pulmonale.

Investigations:

1. Investigations for diagnosis of Schistosomiasis:

- Stool & urine analysis : for Schistosoma ova.
- Sigmoidoscopy & biopsy
- Serology & skin tests : for Schistosoma antibodies.
- Barium enema: for Schistosoma polyps in the colon.
- Blood picture : for anemia & eosinophilia.

2. Investigations for diagnosis of complications : e.g. liver imaging for hepatic schistosomiasis.

Treatment:

1. Treatment of active Schistosomal infection : anti-bilharzial drugs especially Praziquantel.
2. Treatment of polyps by endoscopic polypectomy.
3. Symptomatic treatment: e.g. treatment of anemia, antidiarrheal drugs



3- Inflammatory bowel disease

Definition:

- This represents an idiopathic chronic inflammatory disease of the GIT.
- There are two varieties of the disease with considerable overlap:
-ULCERATIVE COLITIS (UC). – CROHN'S DISEASE (CD).

Etiology:

The exact cause is not known, but there are theories:

1. Genetic factors: the evidence is
 - Patients have close relatives with the disease, and there is high concordance in identical twins.
 - HLA-DR3 is associated with severe ulcerative colitis.
 - Patients with IBD & HLA-B27 commonly develop ankylosing spondylitis
2. Immunological : presence of associated autoimmune manifestations e.g. SLE & thyroiditis.

3. Latrogenic: prolonged use of NSAIDs.
4. Associated with low residue, high refined sugar diet.
5. Infection: unproven theory).

Incidence:

- Age: young (20-30 years).
- Sex: more in females.
- Special habits: Smoking : protects against UC, BUT is associated with CD.

<u>Ulcerative colitis</u>	<u>Crohn's disease</u>
Definition: A chronic inflammatory disorder of the mucosa of the colon. Pathology: 1. <u>Site</u>: The inflammation is continuous and involves the mucosa only. <u>The site of the lesion may be:</u> <i>Proctitis</i>: affection of the rectum only. <i>Left-sided colitis</i>: affection of the rectum + distal colon. <i>Total colitis</i>: affection of the rectum + whole colon. Inflammation of terminal ileum may occur (back wash ileitis) 2. <u>Macroscopic picture</u>: ➤ Multiple ulcerations. ➤ In long standing conditions the bowel becomes shortened with pseudopolyps within the areas of atrophy. 3. <u>Microscopic picture</u>: The inflammatory process affects the mucosa only Infiltration by chronic and acute inflammatory cells There is loss of goblet cells with crypt abscesses Clinical Picture: A. <u>INTESTINAL MANIFESTATIONS</u>: <u>Age</u> : any age but frequently 20-40 years <u>Sex</u> : females > males A. Acute attack: ▪ Proctitis: Bleeding per rectum: is the most important feature. ▪ Proctosigmoiditis: Diarrhoea with mucus & may occur more than 10 times daily, tenesmus, lower abdominal colicky pain. B. Chronic disease: ▪ After recovery from the acute	Definition: A chronic inflammatory disorder which affects: Any part of the GIT from the mouth to the anus. The whole thickness of the bowel wall is inflamed. Pathology: 1. <u>Site</u>: terminal ileum, colon, ileum & jejunum 2. <u>Macroscopic picture</u>: ➤ The entire wall of the bowel is thickened and edematous ➤ There are deep ulcers that appear as linear fissures, thus the mucosa in between is described as cobble-stone. ➤ Fistula is common between adjacent loops of the bowel or with other organs e.g. the urinary bladder. 3. <u>Microscopic picture</u>: ➤ There is infiltration of the whole bowel wall. ➤ Non caseating granulomas may be detected Clinical Picture: A. <u>INTESTINAL MANIFESTATIONS</u>: ▪ Acute colicky pain & tenderness: in the right iliac fossa (simulating acute appendicitis). ▪ Mass: in the right iliac fossa. ▪ Malabsorption syndrome: diarrhea, steatorrhea, loss of weight. ▪ Intestinal obstruction. ▪ Colonic CD: features similar to UC.

attack, the patient returns to a normal bowel habit. - Then the disease runs a chronic intermittent course characterized by: Acute exacerbations: precipitated by NSAIDs or Nervous stress. <u>B. EXTRA-INTESTINAL MANIFESTATIONS:</u> - General :Fever + constitutional manifestations. - Nutritional: Anemia + manifestations of vitamin deficiency. - Eye: Conjunctivitis + iridocyclitis. - Skin: Erythema nodosum + pyoderma gangrenosum + vasculitis. - Arthritis. - Gall stones, sclerosing colangitis and colangiocarcinoma - Autoimmune hepatitis, fatty liver. - Kidney: Oxalate stones. - Interstitial pulmonary fibrosis. Complications: - Hemorrhage - perforation - Megacolon: acute (toxic) dilatation of the colon. - Malignancy: increased risk in cases of "total colitis of 8 years duration" Investigations: <u>Stool analysis & culture:</u> - To exclude an infective colitis. - Presence of :WBCs. RBCs due to inflammation. <u>Colonoscopy:</u> - Lesion : diffuse inflammation, friable mucosa, multiple ulcerations. - Biopsy: multiple CRYPT ABSCESSSES - To detect areas of dysplasia as screening for carcinoma. <u>Barium enema:</u> - Lead – pipe appearance: loss of haustrations + narrowing of the lumen. <u>Blood tests :</u> - Anemia, Leucocytosis. - Abnormal liver function tests. - Increased ESR and C-reactive protein.	<u>B. EXTRA-INTESTINAL MANIFESTATIONS:</u> - Same as Ulcerative Colitis. Complications: Same as Ulcerative Colitis, BUT: - Malignancy: is less common in CD. - Fissures, Fistulas, Abscesses: are more common in CD. Investigations: <u>Stool analysis & culture:</u> - To exclude an infective colitis. - Presence of WBCs, RBCs due to inflammation. <u>Colonoscopy & Ileoscopy:</u> - Lesion : diagnoses the characteristic pathology & detects colonic affection. - Biopsy : differentiates between CD and UC. <u>Barium enema:</u> To detect colonic affection. <u>Barium follow-through:</u> - Deep ulcerations. - String sign : areas of narrowing - SKIP LESIONS: areas of normal bowel in between the lesions. <u>Blood tests:</u> - Anemia, Leucocytosis - Abnormal liver function tests
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Treatment: A. <u>MEDICAL TREATMENT:</u> 1. ACUTE ATTACK: a. Steroids: "the most important for the acute attack" ▪ Prednisone, orally 1 mg /kg /day : in acute attacks. ▪ Hydrocortisone IV 100 mg / 6 hours : in severe attacks (for 5 days). ▪ Hydrocortisone retention enema: in severe proctitis. b. Salicylates : ▪ Sulphasalazine (sulpha + salicylates): 1 gm / 6hours orally. ▪ Mesalamine (5 – aminosalicic acid) : 1 gm / 6 hours orally. c. Symptomatic treatment: ▪ Anti-diarrhea agents: for severe diarrhea. ▪ Antibiotics : for severe secondary bacterial infection. 2. RASISTANT CASES: ▪ Immunosuppressive: Azathioprine & Cyclosporine maybe beneficial. 3. MAINTENANCE: ▪ Sulphasalazine : 2 gm orally daily for 2 years. ▪ Mesalamine : 2 gm orally daily for 2 years. <u>Medical treatment of Fulminant UCs:</u> ▪ IV fluids - Blood transfusion - nutritional support. ▪ IV cyclosporine or infliximab - IV methylprednisolone 60 mg /day ▪ Antibiotics.	Treatment : A. MEDICAL TREATMENT: 1. <u>ACTIVE ILEITIS:</u> Prednisone : orally 1 mg /Kg / day. Immunosuppressive : Azathioprine & Cyclosporine may be beneficial in resistant cases. 2. <u>ACTIVE COLITIS :</u> Prednisone : orally 1 mg /Kg / day. Immunosuppressive : Azathioprine & Cyclosporine may be beneficial in resistant cases. Sulphasalazine (sulpha + salicylates) : 1 gm / 6 hours orally. ➤ Metronidazole : 800 mg tds orally. B. SURGICAL TREATMENT: Indications: 1.Failure of Medical ttt. 2.Development of severe complications. 3.Failure of growth in children Operation : ♦ Standard : Limited resection with end to end anastomosis. ♦ In severe colitis : Proctocolectomy with permanent ileostomy.
B. <u>SURGICAL TREATMENT :</u> ➤ <u>Indications:</u> ♦ Failure of Medical treatment. ♦ Development of severe complications. ♦ Total colitis for more than 10 years: because of high cancer risk. ➤ <u>Operation:</u> the standard operation is "Proctocolectomy with permanent ileostomy"	

4-Irritable bowel Syndrome



Definition: A group of abdominal symptoms for which no organic cause can be found.

Etiology :

- Motility disturbances: increased or decreased strength of colonic motility.
- Psychological disturbances : evidence of associated stress, depression, or anxiety.
- Abnormality of gut flora.

Clinical Types:

1. Spastic colitis : chronic abdominal & constipation.
2. Diarrhea: chronic intermittent watery diarrhea.
3. Alternating type: alternating constipation & diarrhea.

Clinical Picture :

1. **PAIN :**

- Character & site : chronic crampy pain in any part of the colon, especially the left iliac fossa.
- Precipitating & relieving factors : pain following meals or stress relieved by passage of flatus or stools.

2. **TENDERNESS:**

- Over the left iliac fossa: and the spastic sigmoid colon may be palpable.

3. **IRRITABLE MOTIONS:**

- Chronic constipation, or diarrhea, or both.
- Ribbon like stools with sense of incomplete evacuation (Tenesmus)

4. **IRRITABLE PERSONALITY:** Features of anxiety or stress may be present.

5. **Non GIT features:**

- Bad breath & unpleasant taste of the mouth.
- Dysmenorrhea and premenstrual tension
- Headache, poor sleeping and fatigue.
- Urinary symptoms e.g. urgency, frequency.

Diagnostic criteria (Rome criteria):

Abdominal pain or discomfort that has two or more of the following :

1. Relieved with defecation
2. Onset associated with change in frequency of stool.
3. Onset associated with a change in form or appearance of stool.

Diagnosis:

- Diagnosis of IBS is essentially a CLINICAL DIAGNOSIS.
- Investigations are done to exclude organic causes:
 - Stool analysis & culture.
 - Sigmoidoscopy & rectal biopsy: in cases of persistent diarrhea.

Treatment :

- **Psychotherapy** : reassurance & tranquillizers.
- **Symptomatic** : High-fiber diet & laxatives for constipation, loperamide for diarrhea, and antispasmodics for pain.
- **Constipation predominant**:
 1. Selective serotonin reuptake inhibitor: paroxetine (Seroxat).
 2. Serotonin receptor agonist: Tegaserod (Zelmac) 6 mg/12 hrs
- **Diarrhea predominant**: tricyclic anti-depressant e.g. amitriptyline (Tryptizole) 10-25 mg

5-Miscellaneous

☒ Megacolon**A. Hirschsprung's disease: congenital****B. Acquired megacolon:**

- Psychogenic megacolon (neglecting the desire)
- Antidepressant drugs.
- hypothyroidism
- Prolonged laxative abuse
- ulcerative colitis
- Scleroderma

Most patients managed by treatment of the cause, prokinetics, subtotal colectomy may be needed.

☒ Polyposis(D.D)**✓ Familial adenomatous polyposis**

It is an autosomal dominant disease characterized by adenomas of the colon (neoplastic polyposis)

- ✓ **Peutz-jeghers syndrome** : (colonic polyps + pigmentation of lips and fingers).
- ✓ **Turcot's** (colonic polyps + malignant central nervous tumors).
- ✓ **Cronkhit-canada syndrome** (colonic polyps + hair loss and hyperpigmentation).
- ✓ **Cowden's disease** (colonic polyps + orocutaneous hamartomas, thyroid tumors).

Familial adenomatous polyposis

It is an autosomal dominant

Pathology: There are 100 or more adenomas and the rectum is usually involved.

Clinical Picture:

- Symptoms usually begin in the mid 30s
- Abdominal pain
- Diarrhea, blood and mucous in stool.
- The polyps appear at adolescence and become malignant within 15 years and the patient usually dies before the age of 40.
- When osteomas of the jaw, sebaceous cysts and colonic polyps are present the condition is called Gardner's syndrome.

Investigations:

- Baenema shows multiple small filling defects throughout the colon.
- Upper GI endoscopy as gastric, duodenal and periampullary polyps are also common.
- All relatives must be examined by annual sigmoidoscopy after the age of 10 years.

Treatment:

- Total colectomy with ileorectal anastomosis with follow up to detect cancer.
- It is safer to remove the rectum also (proctocolectomy and ileostomy).

☒ **Diverticular disease of the colon**

Pathology:

- There is pouches of mucosa extrude through the muscular wall through weakened areas near blood vessels form diverticula, common in sigmoid colon
- Diverticulitis occurs when faeces obstruct the neck of the diverticulum → stagnation → bacterial multiplication → inflammation. This may lead to perforation, abscess, fistulae and peritonitis.

☒ **Intestinal obstruction**

	Mechanical	A dynamic (paralytic ileus)
Aetiology	<u>1-luminal:</u> ▪ Colonic tumors ▪ Gall stone ileus ▪ Foreign bodies ▪ Meconium ileus <u>2-Mural:</u>	• Recent abdominal surgery. • Electrolyte disturbance especially hypokalemia • Chemical or bacterial peritonitis.

	- Stricture - Intussusception - Hematomas from trauma 3-Extramural: - Adhesions from prior surgery - Strangulated hernia. - Volvulus. - Metastatic tumours. - Endometriosis	Severe intra abdominal inflammation, e.g. pancreatitis.
c/p	Abdominal colic, vomiting and constipation without passage of flatus with increase gut sounds on examination	Abdominal distension with diminished bowel sounds
Investigations:	X-ray abdomen (Erect position) , CT scan can localize	X-ray abdomen shows diffuse diffuse intestinal gas.
Treatment	IV fluids. surgery	Treatment of the cause



GIT BLEEDING

1-HEMATEMESIS

Definition: Vomiting of bright blood.

Etiology:

A. Oesophageal causes:

1. Esophageal varices.
2. GORD: causes esophageal erosions.
3. Cancer esophagus
4. Mallory – Weiss syndrome: tear of the oesophago – gastric junction
5. Rupture of aortic aneurysm into the oesophagus.

B. Gastro – duodenal causes:

1. Acute gastritis: drugs, especially aspirin, NSAIDs, corticosteroids.
2. Chronic peptic ulcer: gastric ulcer OR duodenal ulcer.
3. Bleeding gastric varices: fundal varices
4. CORD : associated with hiatus hernia
5. Cancer stomach and cancer ampulla of Vater.
6. Peutz – Jeghers syndrome: GI polyposis & mucocutaneous pigmentation
7. Angiodysplasia of the stomach

C. Small intestinal causes:

1. Angiodysplasia of the small intestine
2. Familial polyposis
3. Cancer: adenocarcinoma
4. Ulcers

D. General causes:

1. Hemorrhagic blood diseases : purpura, hemophilia & leukemia
2. Hemorrhagic fevers : plague.

E. False hematemesis:

1. Vomiting of ingested blood coming from : bleeding nose, mouth OR pharynx,

Differential Diagnosis :

- Differentiation from hemoptysis: (refer to the chest).
- Differentiation from false hematemesis : by local examination
- DD of the cause of hematemesis through:
 1. Clinical picture
 2. Investigations
 - Upper endoscopy & enteroscopy
 - Barium meal & follow through.
 - Liver function tests & abdominal ultrasonography

2-Melena

Definition : passage of black tarry stools due to the presence of digested blood.

Etiology: Same causes of hematemesis

Differential diagnosis:

- DD of dark stools:
 1. Intake of iron
 2. Hemolytic anemia
- DD of the cause of hematemesis
 - Management of upper GIT hemorrhage:

- **History** and physical examination, Add **Venous access**.
- **Assess severity** of bleeding by pulse, blood pressure, presence of shock and insure patent airway in severe bleeding or come.
- **Lab** for hematocrit, hemoglobin, creatinine and blood urea nitrogen, liver function tests and random blood sugar
- **Replacement of blood** loss by IV fluids and blood transfusion.
- Nasogastric lavage
- **Upper endoscopy**: should be done within 24 hours in most cases, It helps in in diagnosis of cause of bleeding and also for therapeutic interventions e.g. band ligation or sclerotherapy for varices, photocoagulation or APC (argon plasma coagulation) or injection of bleeding ulcers, polypectomy ..etc.
- **Drug therapy**: like IV H₂ –receptor antagonists and Proton Pump inhibitors (PPI's), Somatostatin and vasopressin can be given as IV infusion if the bleeding is difficult to stop.
- **surgery** in : uncontrolled or prolonged bleeding, severe rebleeding, aortoenteric fistula. For intractable variceal bleeding, consider transjugular intrahepatic porto systemic shunt (TIPS).

3-Bleeding per – rectum

Etiology:

- | | |
|---------------------------------|-----------------------------|
| ▪ Piles: the most common cause. | ▪ Colonic polyps : familial |
| ▪ Cancer rectum OR colon | ▪ IBD (UC) |
| ▪ Bacillary dysentery | ▪ Anal fissure |
| ▪ Diverticulitis | |
| ▪ TB of the colon | |
| ▪ Bacillary dysentery | |

treatment : treatment of the cause



symptomatology 1-constipation

Definition :

Presence of two or more of the following :

1. Infrequent stools: < 3/week
2. Hard stools > 1/4 times of defecation
3. Incomplete evacuation : > 1/4 times of defecation
4. Straining > 1/4 times of defecation
5. Manual maneuvers as digital evacuation and support of pelvic floor > 1/4 times of defecation

physiology of defecation :

➤ **Faecal propulsion:**

- * In the inter-defecation period: faeces are transported from the caecum to the sigmoid.
- * In the defecatory period: faeces are transported from the sigmoid to the rectum

➤ **Faecal expulsion:**

- * Sensory phase : Rectal distension triggers defecatory urge
- * Voluntary phase: Straining increases the abdominal pressure Levator ani muscle maintains this pressure Rectal distension causes reflex relaxation of anal sphincters.

Causes of constipation :

❖ **Defective propulsion :**

A. Motility disorder:

1. Colonic inertia : hypomotility due to
 - a. Poor dietary intake of fiber
 - b. Prolonged rest in bed.
 - c. Metabolic: pregnancy, myxoedema, D.M & hypercalcaemia
 - d. Psychogenesis: depression, neurosis & schizophrenia
2. Constipation dominant IBS : due to in-coordination of motility.
3. Paralytic ileus:- hypomotility e.g. postoperative & hypokalemia.
4. Hirshsprung's disease: amotility of segment of the colon.

B. Obstruction :

1. **Intrinsic** : e.g. cancer, faecal impaction or strictures
2. **Extrinsic** : Huge mass in the abdomen.

❖ **Defective expulsion :**

A. Recto-anal dysfunction:

1. **Rectal hyposensitivity:**

Due to marked distension of the rectum, caused by the neglect of the act of defecation (Dyschezia), as in :

- Painful anal conditions e.g. anal fissure

- Professional obligations e.g. early hours jobs
- Unsuitable W.C. conditions e.g. during traveling

2. **Absent recto- anal inhibitory reflex** : Due to autonomic neuropathy

❖ **Pelvic floor dysfunction:**

1. Perianal descent syndrome : weakness of levator ani, as in repeated birth injuries, will lead to inability to maintain a high abdominal pressure.
2. Spastic floor syndrome (anismus): spasm of anal sphincter instead of its relaxation. It is due to history of child abuse or painful anus.

Approach to diagnosis:

★ **History and physical examination:**

- History: dietary habits, fluid intake , ignoring urge to defecate, medications and other symptoms suggestive for systemic disease.
- PR examination: to detect piles, anal fissures

Investigation:

1. Plain x-ray: for intestinal obstruction & megacolon.
2. Barium enema: for organic destruction.
3. Colonoscopy: when alarming signs of structural diseases are present e.g. new onset constipation, anemia ,family history of cancer colon., weight loss, bleeding in rectum or occult blood in stool and
4. Radiologic colonic transit time
5. Defecography.
6. Manometry: colonic & anorectal

Treatment:

1. Treatment of cause
2. If no cause is found
 - Patient education : for proper bowel habits
 - Diet : wheat bran is one of the best laxatives and plenty of fluids
 - Laxatives:
 - Bulk laxatives (fiber based laxatives)
 - Osmotic laxatives (lactulose).
 - Stimulant laxatives (as senna)
3. Tegaserod (serotonin agonist) in IBS.
4. Surgical treatment : rarely needed as in megacolon.



2-Diarrhea

Definition : Change of the normal bowel habits in the form of :

- Increase in frequency or amount or Fluid consistency
- Feces exceeding 200 gm/day when the dietary fiber content is low

Pathogenesis:

1. Osmotic diarrhea:

- Due to : Presence of high concentration of non – absorbed hypertonic substances in intestine which shift fluid to intestine leads to loose stools.
- Examples : lactulose
- Diarrhea stops when the patient is fasting

2. Secretory diarrhea:

- Due to : active intestinal secretions of fluid & electrolytes
- Examples: Enterotoxins, cholera & E.coli.
- Diarrhea does not stop when the patient is fasting.

3. Inflammatory diarrhea: "Mucosal destruction"

- Due to: Damage to the intestinal mucosa, causing loss of fluid & blood
- Examples: Bacillary dysentery. Ulcerative colitis.
- Diarrhea partially improves when the patient is fasting.

4. Abnormal motility :

- Due to: Hypermotility which causes defective absorption
- Examples: Throtoxicosis & post-vagotomy.
- Diarrhea partially improves when the patient is fasting.

A- Acute diarrhea

Etiology:(A i d)

A. Infection:

A.Bacterial	B.Viral	C. protozoal	D. Helminthic
1. Shigella 2. Cholera 3. Coli. 4. Food poisoning: 5. Salmonella, staph & clostridium	1. <u>Rota virus:</u> watery diarrhea & vomiting in children & infants 2. <u>Norwalk virus:</u> diarrhea & vomiting in older children & adults. 3. <u>Echo virus.</u>	1. Ent. Histolytica 2. Giardia Lamblia 3. Malignant malaria 4. Balantidium coli.	1. Ascaris 2. Ankylostoma 3. Strongyloides stercoralis

B. Inflammation : Appendicitis, Ischemic colitis

C. Drugs & Toxins : laxative ,arsenic, lead, some types of mushrooms

D. DIETARY:Excessive cellulose&Food allergies&Unripe fruits, spices

E. Anxiety& nervousness : Exaggerated gastro – colic reflex: e.g. EXAMS!!

Investigations:

1. Stool analysis and culture
2. Sigmoidoscopy: in bloody diarrhea not improving in 10 days

Treatment:

1. Treatment of the cause
2. Fluid replacement
3. Loperamide (2 mg after loose stool) in severe cases as it impairs the clearance of any pathogen in the bowel.

B- Chronic diarrhea

Definition: Diarrhea persisting for more than four weeks

Etiology:

A. Diseases of the colon:

1. Amoebic colitis
2. AIDS: opportunistic infections.
3. Bilharzial colitis
4. Cancer colon
5. IBD
6. Diverticulosis

B. Diseases of the small intestine same causes of intestinal malabsorption**C. Endocrinal causes:**

1. Diabetic neuropathy
2. Thyrotoxicosis
3. Addison's disease
4. Gastrinoma: "Zollinger – Ellison syndrome"
5. Verner – Morrison syndrome : Pancreatic cholera"

D. Drugs:

1. Antibiotics: especially clindamycin which causes pseudomembranous colitis.
2. Purgative abuse

E. Miscellaneous:

1. Obstructive jaundice
2. Pellagra
3. Vitamin B₁₂ deficiency

Diagnosis:

*** History and physical examination:**

1. Watery stool: secretory diarrhea
2. Greasy stool: fatty diarrhea
3. Sensitivity to milk (lactose intolerance), or to wheat (celiac disease)

4. Neuropathy: DM
5. Arthritis : inflammatory bowel disease.

Therapeutic tests:

1. Diet free of milk products → improvement of diarrhea in lactose intolerance
2. Diet free of wheat → improvement of diarrhea in caeliac disease.
3. Metronidazole for giardiasis.

Treatment : As acute diarrhea

3- Tenesmus

Definition: A disease characterized by:

- During defecation: painful straining.
- After defecation: sense of incomplete rectal evacuation + persistent desire to defecate.

Causes:

- ★ Irritable bowel syndrome
- ★ Inflammatory bowel disease : e.g. Ulcerative Colitis, Crohn's Disease
- ★ Infective colitis : e.g. Bacillary Dysentery
- ★ Rectal prolapsed
- ★ Rectal carcinoma
- ★ Causes of Dysentery

4- Epigastric pain

❖ Gastro- Intestinal Causes:

A. Oesophageat	B. Gastro-duodenal	C. Intestinal	D. Gall bladder
♦ Oesophageal spasm ♦ GORD & oesophagitis. ♦ Hiatus hernia	♦ Gastritis ♦ Peptic ulcer	♦ Ankylostoma infestation ♦ Intestinal tuberculosis ♦ Intestinal obstruction	♦ Cholecystitis ♦ Gall stones ♦ Biliary dyskinesia ♦ Post-cholecystomy syndrome
E. Pancreatic causes			
♦ Carcinoma of the pancreas , Acute pancreatitis			

5-Vomiting

Causes:

A. Reflex Causes:

1. **Naso-pharyngeal causes:** F.B., Inflammation, tumour & induced vomiting.
2. **Oesophageal causes:** Achalasia, stricture & carcinoma.
3. **Gastric causes:** Acute & chronic gastritis, Peptic ulcer, cancer & pyloric obstruction.
4. **Intestinal causes:** Acute intestinal obstruction., appendicitis.
5. **Hepatic & biliary causes:** Acute cholecystitis & biliary colic, Acute viral hepatitis.
6. **Pancreatic causes:** Acute pancreatitis.
7. **Renal causes:** Renal colic, Acute pyelonephritis.
8. **Cardiac causes:** Myocardial infarction & digitalis toxicity.
9. **Ocular causes:** Acute glaucoma
10. **Gynaecological causes:** Dysmenorrhoea

B. Central causes:

1. Psychic : small, slight or taste
2. Hysterical
3. Medullary lesions
4. Archi – cerebellar lesions
5. Vestibular lesion as Menier's disease
6. Migraine
7. Centrally acting emetic drugs morphia

C. Causes acting centrally & peripherally:

1. Uraemia
2. Hyper & hypoglycaemia
3. Acidosis & alkalosis
4. Hyponatraemia & hypo or hypetkalaemia .
5. Addison's disease & hyperparathyroidism.

Diagnosis:

1. History :

- Age & sex
- Time of vomiting
- Associated nausea & abdominal pain (reflex)
- Associated headache & blueing of vision (++ I.C.T.)
- Associated vertigo & deafness (vestibular)
- Of biliary or renal colic & food intake

2. Examination of vomitus:

- Odour: foul in malignancy & intestinal obstruction
- Mucus: chronic gastritis.
- Bile : persistent vomiting below ampulla of Vater
- Blood : ulcer, cancer, or "Mallory – Weiss syndrome"
- F.B. : Ascaris or gall stones

3. Examination of different systems:

Special Investigations: As x-ray, gastroscopy, blood urea....etc

Treatment:

- Treatment of the cause.
- Correct dehydration & electrolyte imbalance
- Domperidon or metoclopramide

6-Dyspepsia

Definition:

Upper abdominal pain or discomfort related to meals. The discomfort may include upper abdominal fullness, early satiety, burning, bloating, nausea or vomiting.

Etiology:

1. **Functional or " non-ulcer dyspepsia "**: this is the most common cause with no structural abnormality. The cause may present interaction of :
 - Increased afferent visceral sensitivity
 - Delayed gastric emptying
 - Psychosocial stressors
2. **Gastrointestinal diseases**: GORD, peptic ulcer, gastric cancer, gastroparesis (in DM), malabsorption syndrome, parasitic infestation.
3. **Biliary tract disease**
4. **Pancreatic disease**
5. **Food or drug intolerance**
6. **Other conditions**: DM, thyroid disease, renal impairment, myocardial ischemia or pregnancy

Diagnosis:

➤ **History:**

- Anxiety, depression
- Associated manifestations: heart burn (GORD), epigastric pain (peptic ulcer).

➤ **Symptoms:**

Warning symptoms that warrant endoscopy or abdominal imaging:

- Weight loss
- Persistent vomiting
- Constant or severe pain
- Dysphagia
- Hematemesis or melena

➤ **Physical examination:**

To detect signs of serious organic disease e.g. cachexia, organomegaly, abdominal mass.

Investigations:

1. **Upper endoscopy** : it is indicated in :

- ★ All patients > 45 years old with recent onset dyspepsia
- ★ All patients with weight loss, dysphagia, recurrent vomiting, evidence of bleeding or anaemia
- ★ Failure of response to therapy

2. **Upper GIT barium radiography**

3. **Abdominal sonography or CT.**

4. **Ambulatory esophageal pH testing.**

Treatment:

1. **Treatment of the cause**

2. **Treatment of functional dyspepsia:**

➤ General measures:

- Reassurance, psychotherapy
- Reduction in coffee, alcohol and caffeine

➤ Pharmacological agents:

- Anti secretory therapy (H₂-blockers or proton pump inhibitors): this may benefit 10-20% of patients especially those with heart burn (reflux-like dyspepsia) or epigastric pain.
- Low dose of antidepressants (nortriptyline 10-50 mg at 3rd time) may moderate afferent visceral sensitivity.
- Prokinetic agents (metoclopramide): improve symptoms in up to 60% of patients



7-Acute Abdomen

Definition: A group of abdominal conditions that cause the patient to be hospitalized within a few hours of the onset of pain in which surgical treatment must be considered intra-abdominal catastrophes, so early surgical consultation should be obtained.

Causes:

- Bowel:
 - ♦ Acute appendicitis
 - ♦ Perforated viscus e.g. peptic ulcer
 - ♦ Diverticular disease
 - ♦ Intestinal obstruction and strangulation
 - ♦ Meckel's diverticulum
 - ♦ Terminal ileitis by bacterial infection (Yersinia)
- Vascular:
 - ♦ Mesenteric occlusion
 - ♦ Rupture aortic aneurysm
- Gynaecological:
 - ♦ Ruptured ectopic pregnancy or ovarian cyst
 - ♦ Acute salpingitis
- Miscellaneous:
 - ♦ Cholecystitis
 - ♦ Acute mesenteric adenitis
 - ♦ Acute pancreatitis

Clinical Picture:

History:

1. Sudden onset of pain suggests perforation of viscus, rupture aneurysm, torsion of ovarian cyst or acute pancreatitis.
2. Back pain suggests pancreatitis, rupture of an aortic aneurysm or renal disease.
3. Inflammatory conditions e.g. (appendicitis) produce a more gradual onset of pain. With peritonitis the pain is continuous and may be made worse by movement.
4. Vomiting may occur with any abdominal conditions but if persistent it suggests intestinal obstruction.
5. Ask about other symptoms as change in bowel habit, urinary symptoms and gynaecological history.

Examination:

1. Abdominal tenderness, muscle guarding and rigidity are signs of peritonitis.

2. Inflammation of pelvic peritoneum may produce tenderness on rectal examination.
3. Absent bowel sounds suggest peritoneal involvement, strangulation or ischemia or ileus.
4. Search for specific S&S according to the cause.

Investigations:

- Blood picture: TLC in inflammatory conditions
- Serum amylase is high in acute pancreatitis
- X-ray to detect air under diaphragm due to perforated viscus or multiple fluid level in intestinal obstruction.
- Sonar for cholecystitis, appendicitis. Spiral CT for pancreatitis, appendicitis
- Laparoscopy

Medical conditions which may mimic acute abdomen :

- | | |
|----------------------------|---|
| • Myocardial infarction | • FMF |
| • Sickle cell disease | • Diabetic ketoacidosis, Addisonian crisis. |
| • Pleurisy | • Gastroenteritis |
| • Vasculitis | • Henoch Schonlein purpura |
| • Irritable bowel syndrome | • Herpes zoster |